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Oxygen Transport

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OXYGEN TRANSPORT includes those processes which move oxygen from inspired air to the sites of intracellular utilization. The product of arterial oxygen content (Ca_{O_2}) and cardiac output ($\dot{Q}_T$) represents the quantity of oxygen available ($\dot{Q}_T \cdot Ca_{O_2}$) each minute. Pulmonary gas exchange, cardiac output, hemoglobin concentration, hemoglobin–oxygen affinity, and oxygen consumption of the body are important determinants of oxygen transport. The purpose of this analysis is to describe graphically the extent to which alterations in the above variables disturb oxygen transport, with particular reference to alterations in the anesthetized patient.

Oxygen Transport System

Pulmonary gas exchange normally produces an arterial oxygen tension (Pa_{O_2}) only minimally lower than the average alveolar oxygen tension (PA_{O_2}).[1] Many factors may promote hypoxemia during anesthesia.[2] One of these factors, right-to-left shunts ($\dot{Q}_S/\dot{Q}_T$), is used below to illustrate the effects of altered pulmonary gas exchange on oxygen transport.

Cardiac output is a major determinant of oxygen transport. The effect of low cardiac output on arterial oxygenation is of special interest to the anesthesiologist. Low cardiac output during general anesthesia may result from pre-existing cardiac disease, effects of anesthetic agents or techniques, hemorrhage or mechanical impairment of the circulation.

Hemoglobin concentration is another major determinant of oxygen transport. Blood oxygen-carrying capacity = 1.38 ml O_2/g hemoglobin. Other factors will reduce blood oxygen-carrying capacity by binding available hemoglobin and preventing its use as an oxygen carrier. As an example, in heavy smokers, carbon monoxyhemoglobin (COHb) may bind 10 per cent of total hemoglobin.

Hemoglobin–oxygen affinity refers to the relationship between hemoglobin oxygen saturation (S_{O_2}) and oxygen tension (P_{O_2}). Hydrogen ions, carbon dioxide, and 2,3-diphosphoglycerate shift the hemoglobin-dissociation curve to the right, favoring release of oxygen. Shifting the upper, relatively flat, hemoglobin-dissociation curve has little effect on the pulmonary uptake of oxygen by hemoglobin.

Oxygen consumption of the body ($\dot{V}_{O_2}$) is influenced by temperature, muscle activity, anesthetic agents and techniques, and pre-existing disease.

Pulmonary gas exchange, hemoglobin–oxygen affinity, and oxygen consumption are discussed in separate papers in this symposium.

Oxygen Transport Model

The quantity of oxygen delivered from the lung each minute:

$$(\dot{Q}_T - \dot{Q}_S) \cdot Cc'_{O_2})$$

when combined with the contribution of venous blood passing from the right to left side of the circulation:

$$(\dot{Q}_S \cdot Cv_{O_2})$$

equals the total amount of oxygen available each minute:

$$(\dot{Q}_T \cdot Ca_{O_2}), \quad \therefore$$

$$\dot{Q}_T \cdot Ca_{O_2} = [(\dot{Q}_T - \dot{Q}_S) \cdot Cc'_{O_2}] + (\dot{Q}_S \cdot Cv_{O_2}) \quad (1)$$

From the Fick equation:

$$Cv_{O_2} = Ca_{O_2} - (\dot{V}_{O_2}/\dot{Q}_T) \quad (2)$$

Substituting the equivalent of Cv_{O_2} from equation 2 into equation 1:

$$Ca_{O_2} = Cc'_{O_2} - \left(\frac{\dot{Q}_S}{\dot{Q}_T} \cdot \frac{\dot{V}_{O_2}}{(\dot{Q}_T - \dot{Q}_S)}\right) \quad (3)$$

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This has been used previously to predict[3] and measure[4] the effects of cardiac output on arterial oxygenation. The increased $\dot{V}_{O_2}$ associated with alkalosis[5,6] may also promote lower arterial oxygen values.[7]

In the examples of oxygen transport that follow, the model is a 70-kg man at sea level with a 5-liter blood volume (arterial 25 per cent and venous 75 per cent). Normal cardiac output = 6 l/min; $\dot{V}_{O_2}$ = 300 ml/min, arteriovenous oxygen content difference

$$(Ca_{O_2} - C\bar{v}_{O_2}) = 50 \text{ ml/l.}$$

Inspired oxygen concentration ($F_{I_{O_2}}$) = 40 per cent, alveolar ventilation is normal, with alveolar carbon dioxide tension ($P_{A_{CO_2}}$) = 40 torr, *p*H = 7.4, respiratory quotient (R.Q.) =0.8. All calculations are based on steady-state conditions. Oxygen transport is concerned with quantities of oxygen. Arterial oxygen content (Ca_{O_2}) is therefore of primary importance, and is used in the examples rather than Sa_{O_2} or Pa_{O_2}. As mentioned previously, $\dot{Q}_S/\dot{Q}_T$ will be used to demonstrate the role of hypoxemia in altering oxygen transport. The effect of uneven alveolar ventilation/pulmonary capillary blood flow ratios ($\dot{V}_A/\dot{Q}_C$) in lowering Ca_{O_2} is minimal or absent when $F_{I_{O_2}}$ is 40 per cent or more.[1] When there is marked inequality in $\dot{V}_A/\dot{Q}_C$, the above assumption may not be true.[8] $\dot{Q}_S/\dot{Q}_T$ is used here to represent the total equivalent contribution of mixed venous blood to the final arterial value.

The effects of cardiac output with different $F_{I_{O_2}}$'s and hemoglobin concentrations are separately illustrated in figures 2 and 3. In each example, $\dot{V}_{O_2}$ and $\dot{Q}_S/\dot{Q}_T$ have a constant value. If, for example, cardiac output is reduced by a third to 4 l/min, increasing $F_{I_{O_2}}$ from 40 to 100 per cent (fig. 2) has a minimal effect on oxygen delivery ($\dot{Q}_T Ca_{O_2}$ increases by 6 per cent, from 800 to 850 ml/min). The separate effects on oxygen delivery and arterial oxygenation† are important to remember: Ca_{O_2} (198 to 212 ml/l), Sa_{O_2} (94.7 to 99.4 per cent), Pa_{O_2} (73 to 208 torr). Failure to consider the lesser effect of increasing $F_{I_{O_2}}$ on oxygen delivery may lead the clinician to overestimate its value in substantially-improving oxygen delivery. If, in addition to a one-third reduction in cardiac output to 4 l/min, hemoglobin is also reduced (fig. 3) by a third to 100 g/l (a value not infrequently seen preoperatively), a more critical value of oxygen delivery is established ($\dot{Q}_T Ca_{O_2}$ = 520 ml/min). Normally, a fourth of the available oxygen is used each minute (300/1,200 = 25 per cent). In the example under discussion Ca_{O_2} = 130 ml/l (13 vol per cent). The extraction of oxygen by the myocardium is normally high (110–130 ml/l). Under the above conditions and with normal myocardial blood flow, the coronary sinus oxygen content will approach zero. If possible, the heart will respond by decreasing oxygen utilization and/or increasing myocardial blood flow. This is but one example of the importance of maintaining oxygen delivery well above the amount of oxygen being consumed by the body each minute. The quantity of oxygen delivered in excess of the amount consumed each minute needed to sustain life

$$Ca_{O_2} = Cc'_{O_2} - \left[\frac{\dot{Q}_S}{\dot{Q}_T} \cdot \frac{\dot{V}_{O_2}}{(\dot{Q}_T - \dot{Q}_S)}\right]$$

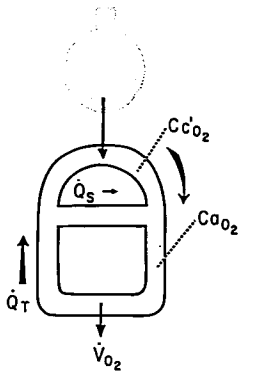

FIG. 1. Oxygen transport model. The oxygen content of the body is constant (steady-state) when volume of oxygen uptake via the lung equals oxygen consumption of the body. Ca_{O_2} depends upon Cc'_{O_2}, $\dot{Q}_S/\dot{Q}_T$, $\dot{V}_{O_2}$ and $\dot{Q}_T$. Oxygen delivery ($\dot{Q}_T \cdot Ca_{O_2}$) in turn depends upon Ca_{O_2} and $\dot{Q}_T$.

† The values of Sa_{O_2} and Pa_{O_2} compatible with the Ca_{O_2} are solved by an iterative technique using a digital computer. The computer program is written in the BASIC language,[9] and makes use of Kelman's equation[10] for the Severinghaus oxygen-dissociation curve.[11] Copies are available to interested readers.

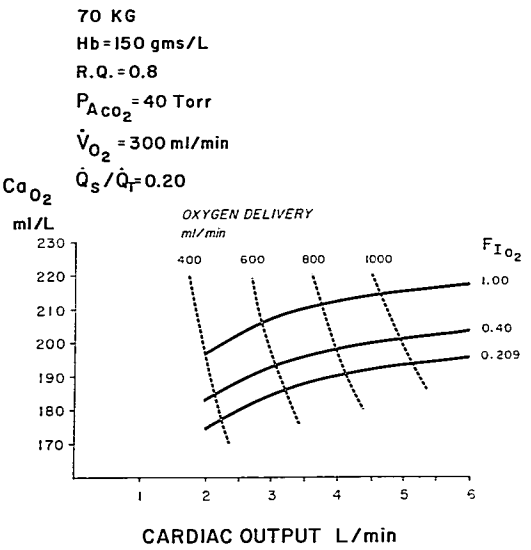

FIG. 2. Effects of cardiac output and inspired oxygen concentration on oxygen delivery (70-kg man). For example, during breathing of 40 per cent O_2, a reduction of cardiac output from 6 l/min to 4 l/min decreases oxygen delivery from approximately 1,200 to 800 ml/min. With a cardiac output of 4 l/min, increasing $F_{I_{O_2}}$ from 40 to 100 per cent O_2 increases oxygen delivery by 50 ml/min. From the illustration, it is obvious that at lower values of cardiac output increasing $F_{I_{O_2}}$ has a decreasing effect on oxygen delivery.

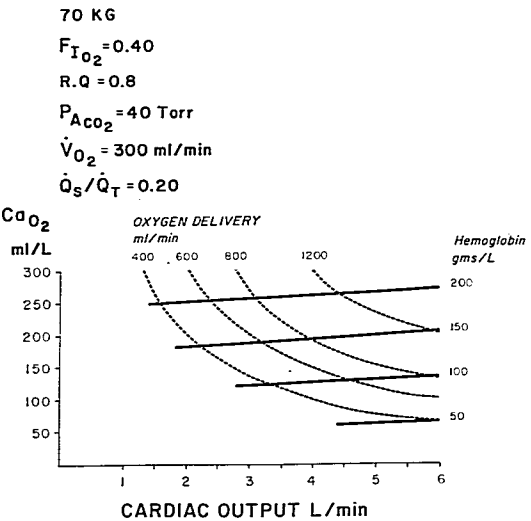

FIG. 3. Effects of cardiac output and hemoglobin concentration on oxygen delivery.[5] For example, with a normal hemoglobin concentration (150 g/l), a reduction in cardiac output from 6 to 4 l decreases oxygen delivery (as in fig. 2) from 1,200 to 800 ml/min. With a cardiac output of 4 l/min, a reduction in hemoglobin from 150 to 100 g/l decreases oxygen delivery from 800 to 520 ml/min.

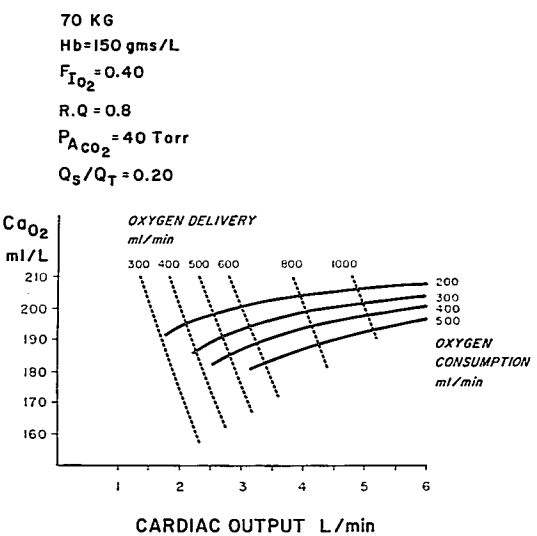

FIG. 4. Effects of cardiac output and oxygen consumption on oxygen delivery. For example, with cardiac output = 4 l/min, increasing $\dot{V}_{O_2}$ from 300 to 400 ml/min decreases Pa_{O_2} from 73 to 64 torr, Sa_{O_2} from 95 to 92 per cent, Ca_{O_2} from 198 to 193 ml/l. Oxygen delivery decreases by only 20 ml/min. The ratio of oxygen consumption/oxygen delivery ($\dot{V}_{O_2}/\dot{Q}_T Ca_{O_2}$) is changed significantly from 38 to 52 per cent. The small decrease in arterial oxygenation is associated with a significant decrease in oxygen delivery.

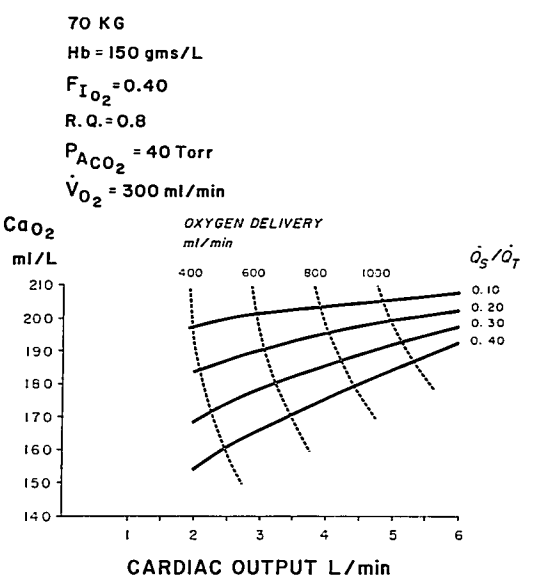

FIG. 5. Effects of cardiac output and $\dot{Q}_S/\dot{Q}_T$ on oxygen delivery. For example, with cardiac output = 4 l/min, an increase of $\dot{Q}_S/\dot{Q}_T$ from 20 to 40 per cent decreases Pa_{O_2} from 73 to 54 torr, while the ratio of $\dot{V}_{O_2}/\dot{Q}_T Ca_{O_2}$ increases only from 38 to 41 per cent. Another example pointing out the need to consider arterial oxygenation and oxygen delivery as entities, each requiring separate attention.

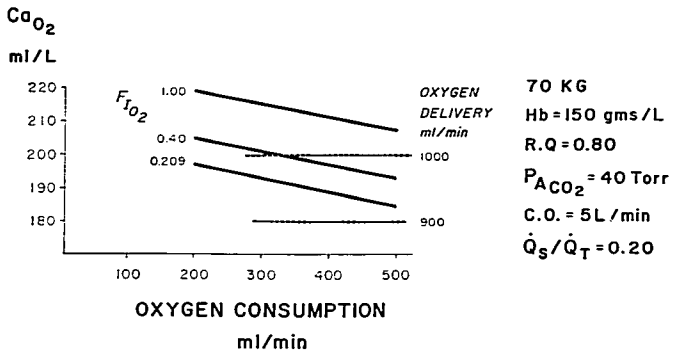

FIG. 6. Effects of oxygen consumption and $F_{I_{O_2}}$ on oxygen delivery (70-kg man). For example, increasing $\dot{V}_{O_2}$ from 300 to 400 ml/min decreases oxygen delivery by 20 ml/min. Increasing $F_{I_{O_2}}$ from 40 to 100 per cent increases oxygen delivery by 60 ml/min.

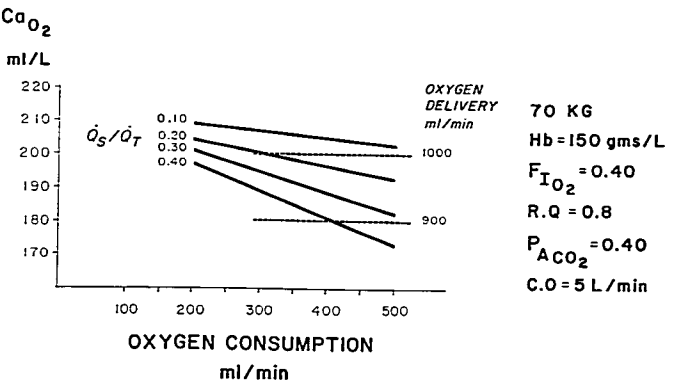

FIG. 7. Effects of oxygen consumption and $\dot{Q}_S/\dot{Q}_T$ on oxygen delivery. For example, the combined effect of increasing $\dot{Q}_S/\dot{Q}_T$ from 20 to 40 per cent and $\dot{V}_{O_2}$ from 300 to 400 ml/min decreases Pa_{O_2} from 82 to 52 ml/min, Sa_{O_2} from 96 to 87 per cent, Ca_{O_2} from 201 to 181 ml/l. $\dot{V}_{O_2}/\dot{Q}_T Ca_{O_2}$ increases from 33 to 44 per cent.

is uncertain. An oxygen delivery equal to twice the amount being consumed would seem to provide an adequate amount, although 600 ml/min ($2 \times \dot{V}_{O_2}$) is only half of normal delivery (1,000–1,200 ml/min).

Increased $\dot{V}_{O_2}$ will decrease oxygen delivery when other factors ($F_{I_{O_2}} = 0.40$, Hb = 150 g/l, $\dot{Q}_S/\dot{Q}_T = 0.20$) remain constant. For example (fig. 4), with cardiac output at 4 l/min, an increase in $\dot{V}_{O_2}$ from 300 to 400 ml/min will reduce $\dot{Q}_T Ca_{O_2}$ by only 3 per cent (790 to 770 ml/min). If hemoglobin were 100 g/l, the same change in $\dot{V}_{O_2}$ would produce a similar decrease in $\dot{Q}_T Ca_{O_2}$ (520 to 500 ml/min). The obviously larger difference in oxygen delivery is controlled by the hemoglobin concentration.

Oxygen delivery will also be influenced by

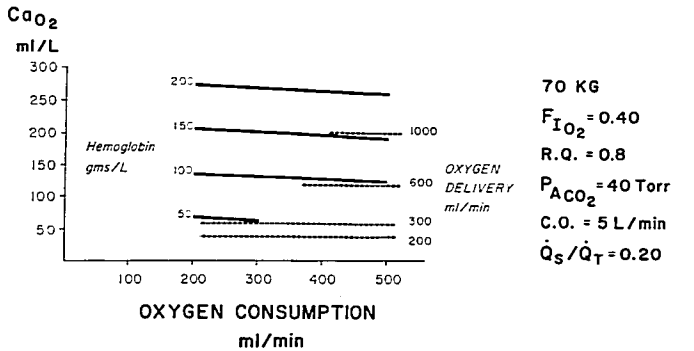

FIG. 8. Effects of oxygen consumption and hemoglobin concentration on oxygen delivery. Increasing $\dot{V}_{O_2}$ has little effect on $\dot{Q}_T Ca_{O_2}$, while hemoglobin has a principal role in altering oxygen delivery.

the magnitude of $\dot{Q}_S/\dot{Q}_T$. From figure 5 it is obvious that increasing values of $\dot{Q}_S/\dot{Q}_T$ significantly alter the value of Ca_{O_2}. Maintenance of normal cardiac output, oxygen consumption, and hemoglobin concentration will prevent a significant decrease in oxygen delivery.

Analyses of the effects of $\dot{V}_{O_2}$ together with $F_{I_{O_2}}$, $\dot{Q}_S/\dot{Q}_T$, and hemoglobin concentration are seen in figures 6, 7 and 8. Hemoglobin = 150 g/l and cardiac output = 5 l/min in figures 6 and 7. Large changes in $F_{I_{O_2}}$ or $\dot{Q}_S/\dot{Q}_T$, when combined with significant changes in $\dot{V}_{O_2}$, have moderate effects on oxygen delivery.

The combined effect of $\dot{V}_{O_2}$ and hemoglobin concentration on oxygen delivery (fig. 8) is related primarily to hemoglobin deficiency.

Oxygen Stores of the Body

Body oxygen stores are located primarily in alveolar gas and the circulating blood volume.[12–15] In normal man, breathing air at sea level, this equals approximately 1,000 ml of oxygen. The quantity of oxygen dissolved in tissues represents a negligible amount compared with the quantity available in lungs and blood.

An increase in $F_{I_{O_2}}$ will obviously increase $P_{A_{O_2}}$ and $P_{a_{O_2}}$. Under normal conditions, increasing $F_{I_{O_2}}$ from 21 to 100 per cent increases $P_{A_{O_2}}$ from approximately 100 to 680 torr. This results in an increase in the dissolved oxygen content in arterial blood of 17 ml/l (from 3 to 20 ml O_2/l arterial blood). Sa_{O_2} increases from approximately 97 to 100 per cent. Under these conditions the increase in oxygen content is due principally to the additional dissolved oxygen.

During anesthesia the magnitude of $\dot{Q}_S/\dot{Q}_T$ will modify the quantity of oxygen transferred from alveolar gas to arterial blood. Again, $\dot{Q}_S/\dot{Q}_T$ is used to represent the factors which promote an arterial value which is lower than alveolar. Figure 9, for example, describes the effects of altering $F_{I_{O_2}}$ from 21 to 40 and 100 per cent on the volumes of oxygen in lungs and arterial and venous blood volumes. This model obviously represents an oversimplification; however, for descriptive purposes, it is useful. Here is a 70-kg man with normal hemoglobin, FRC, ventilation, cardiac output and oxygen consumption, with $\dot{Q}_S/\dot{Q}_T$ equal to 20 per cent of the cardiac output. As $F_{I_{O_2}}$ increases, the magnitude of changes in the oxygen content of the arterial and venous blood volume can be seen. Changes in FRC are now recognized as significant contributors to alterations in body oxygen stores, and maneuvers to prevent a decrease or actually increase FRC, such as positive end-expiratory pressure (PEEP), are now being used clinically.

A primary determinant of oxygen stores of the body, of primary concern to the anesthesiologist, is the hemoglobin concentration of

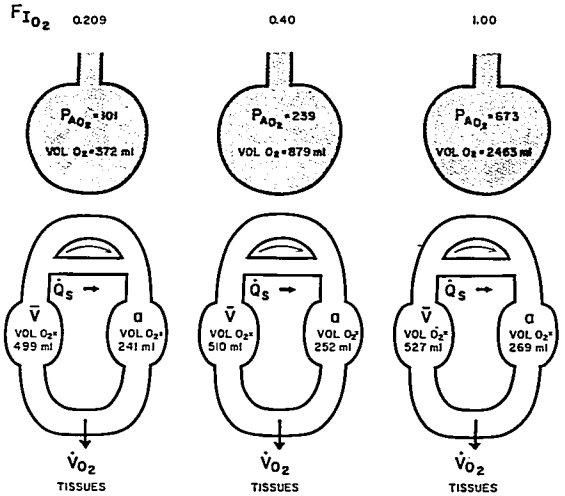

FIG. 9. Effects of $F_{I_{O_2}}$ on oxygen content of the body (70-kg man). Oxygen contents of the body during breathing of 21, 40, and 100 per cent O_2 are approximately 1,100, 1,600 and 3,100 ml.

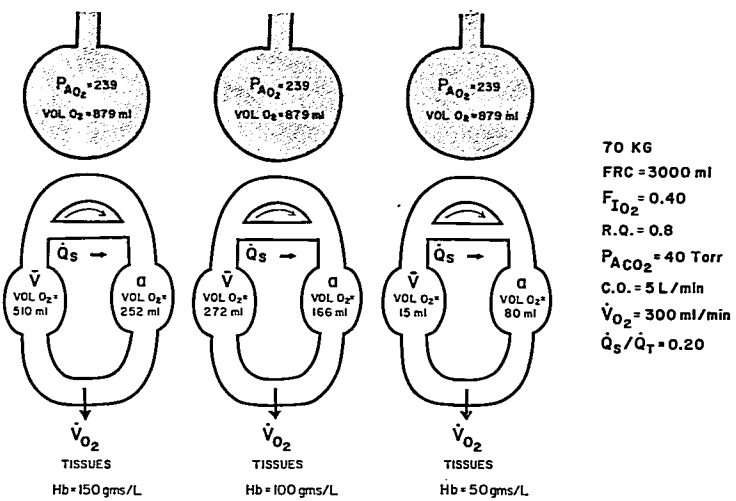

FIG. 10. Effects of hemoglobin concentration on oxygen content of the body. Oxygen contents of the body with hemoglobin 150, 100, and 50 g/l are approximately 1,600, 1,200 and 900 ml, while ratios of $V_{O_2}/\dot{Q}_T Ca_{O_2}$ are 300/1,000, 300/670, and 300/320.

blood. In figure 10 the model patient is breathing 40 per cent oxygen, with the variables the same as in the previous example. The quantity of oxygen available in the lung remains constant, and the effects of changes in blood hemoglobin concentration from normal conditions to marginal and severe anemia are depicted. If all the variables remain constant, then when hemoglobin is reduced from 150 to 50 g/l, the quantity of oxygen in the circulating blood decreases by more than 90 per cent of the amount normally available. These changes are predicated on the basis of normal cardiac output and oxygen consumption. The model of severe anemia (Hb = 50 g/l) is not compatible with survival (fig. 10), because oxygen delivery is only 20 ml/min above $\dot{V}_{O_2}$. This figure is included to point out the necessity of considering all aspects of oxygenation when attempting to achieve optimal conditions for survival.

Summary

The purpose of this paper has been to illustrate in graphic form the relationships which control oxygen delivery. Blood hemoglobin concentration and cardiac output should receive primary attention in therapy aimed at maintaining oxygen transport. Other factors, such as inspired oxygen concentration, oxygen consumption, alveolar ventilation, and factors promoting arterial hypoxemia, also modify oxygen transport. The importance of blood hemoglobin concentration in maintaining adequate oxygen transport cannot be overemphasized.

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Erratum

An error appeared in the article, "Improved Brachial Plexus Blockade with Bupivacaine Hydrochloride and Carbonated Lidocaine," by P. R. Bromage and M. Gertel, in the May issue (ANESTHESIOLOGY 36:479–487, 1972). In table 1 on page 480, the mean duration of blockade for Group 1 (1 per cent lidocaine hydrochloride) should read "196 minutes," instead of "296 minutes."